Amendments to the Specification:

Please replace the paragraph beginning at the bottom of page 2 and extending onto page 3 with the following amended paragraph:

The mtDNA codes for 13 essential OXPHOS polypeptides, 22 tRNA genes, and a 12S and 16S rRNA gene. In addition, the mtDNA CR encompasses the light (L)- and heavy (H)-strand promoters (P_L and P_H); their mitochondrial transcription factor A (mtTFA) binding sites; the downstream conserved sequence blocks (CSB) I, II, and III; and the origins of H-strand replication (O_{H1} and O_{H2}). Recently, tissue-specific, mtDNA CR mutations have been discovered to accumulate with age. A T414G transversion in the mtTFA binding site of P_L accumulates in cultured skin fibroblasts and can be detected at low levels in skeletal muscle, but not in brain, using applicant's sensitive protein nucleic acid (PNA)-clamping polymerase chain reaction (PCR) method. In addition, the A189G and T408A CR mutations accumulate with age in skeletal muscle and a T150C mutation accumulates in white blood cells. However, to date no specific, somatic, mtDNA CR mutations have been reported for normal or AD patient brains. However, specific mtDNA CR mutations have been found to accumulate with age in particular tissues. For example, a T to G transversion at np 414 (T414G) was found to accumulate with age in human skin fibroblasts (Michikawa et al, 1999, *Science* 286:774-779) and an A189G and a T408A mutation were observed to accumulate in skeletal muscle (Wang et al, 2001, *PNAS* 98:4022-4027). However, the T414G mutation could not be detected in normal brain using a sensitive protein nucleic acid (PNA)-clamping polymerase chain reaction (PCR) technique (Murdock et al. 2000~~-2002~~, *NAR* 28:4350-4355).

Please replace the first full paragraph on page 10 with the following amended paragraph:

Additional somatic mtDNA CR mutations were identified by PCR-amplification of the mtDNA CR between nucleotide pairs (nps) 16527 and 636, cloning and sequencing as shown in Figure 1. Frontal cortex genomic DNA was extracted using the pure gene kit (Gentra system) and the CR amplified using the primers np 16527-16546 (5'-CCT AAA TAG CCC ACA CGT TC-3')[SEQ ID NO. 1] and np 617-636 (5'-TGA TGT GAG CCC GTC TAA AC-3') [SEQ ID NO. 2] together with high fidelity Epicentre failsafe Taq DNA polymerase. The desired PCR fragments were purified by agarose gel electrophoresis, extracted using the NucleoTrap gel kit

(Clontech), cloned using the TOPO TA cloning protocol (Invitrogen), and the desired plasmids purified by the mini-preparation. Plasmid DNAs were cycle sequenced using BigDye dideoxy chain terminator chemistry (Applied Biosystem) on an ABI 3100 capillary sequencer, with the sequencing results analyzed using "Sequencer v4.0.5" (Gene Code Corporation).

Please replace the second full paragraph on page 10 with the following amended paragraph:

To determine the ratio of mtDNA L-strand to H-strand transcripts, total RNA was extracted from the cortex tissue using TRIZOL (Gibco-BRL system) and the L-strand, ND6, mRNA and H-strand, ND2, mRNAs were reverse transcribed and quantified by quantitative real time (qRT)-PCR. ND6 was amplified using forward primer np 14260- 14279 (5'-ATC CTC CCG AAT GAA CCC TG-3') **[SEQ ID NO. 3]** and reverse primer np 14466 - 14485 (5'-GAT GGT TGT CTT TGG ATA TA-3') **[SEQ ID NO. 4]**. ND2 mRNA was amplified using the using the same primes as employed to determine the mtDNA/nDNA ratio[25].